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AND CURRENT DISCOVER FILE IS DATED 13 JUNE 2005

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FILE 'USPATFULL' ENTERED AT 16:27:16 ON 24 OCT 2005  
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FILE COVERS 1971 TO PATENT PUBLICATION DATE: 20 Oct 2005 (20051020/PD)

FILE LAST UPDATED: 20 Oct 2005 (20051020/ED)

HIGHEST GRANTED PATENT NUMBER: US6957446

HIGHEST APPLICATION PUBLICATION NUMBER: US2005235390

CA INDEXING IS CURRENT THROUGH 20 Oct 2005 (20051020/UPCA)  
ISSUE CLASS FIELDS (/INCL) CURRENT THROUGH: 20 Oct 2005 (20051020/PD)  
REVISED CLASS FIELDS (/NCL) LAST RELOADED: Aug 2005  
USPTO MANUAL OF CLASSIFICATIONS THESAURUS ISSUE DATE: Aug 2005

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>>> classifications, or claims, that may potentially change from <<<  
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This file contains CAS Registry Numbers for easy and accurate substance identification.

```
=> s FC(w)gamma(w)RIIa
  50010 FC
  19577 FCS
  64972 FC
    (FC OR FCS)
  228507 GAMMA
    838 GAMMAS
  228578 GAMMA
    (GAMMA OR GAMMAS)
  252 RIIA
    1 RIIAS
  252 RIIA
    (RIIA OR RIIAS)
L1    212 FC(W)GAMMA(W)RIIA
```

=> d kwic

L1 ANSWER 1 OF 212 USPATFULL on STN
SUMM . . . Fc $\gamma$ RIC and Fc $\gamma$ RIC) are clustered in region 1q21.1 of the long arm of chromosome 1; the genes encoding Fc $\gamma$ RII isoforms (Fc $\cdot$ gamma.RIIA, Fc $\gamma$ RIIB and Fc $\gamma$ RIIC) and the two genes encoding Fc $\gamma$ RIII (Fc $\gamma$ RIIIA and Fc $\gamma$ RIIIB) are all clustered in region 1q22. These different.

DRWD . . . FIG. 16A shows the binding of immune complexes using different antigen-antibody pairs to recombinant GST fusion protein of the Fc $\cdot$ gamma.RIIA receptor  $\alpha$  subunit. FIG. 16B shows the binding of the same antigen-antibody pairs to the GST fusion protein of the . . .

DRWD . . . the different Fc $\gamma$ Rs. Binding of alanine variants at residues in the CH2 domain of anti-IgE E27 IgG1 are shown to Fc $\cdot$ gamma.RIIA, Fc $\gamma$ RIIB, and Fc $\gamma$ RIIIA. Type 1 abrogates binding to all three receptors: D278A (265 in EU numbering). Type 2 improves binding to Fc $\cdot$ gamma.RIIA and Fc $\gamma$ RIIB, while binding to Fc $\gamma$ RIIIA is unaffected: S280A (267 in EU numbering). Type 3 improves binding to Fc $\cdot$ gamma.RIIA and Fc $\gamma$ RIIB, but reduces binding to Fc $\gamma$ RIIIA: H281A (268 in EU numbering). Type 4 reduces binding to Fc $\cdot$ gamma.RIIA and Fc $\gamma$ RIIB, while improving binding to Fc $\gamma$ RIIIA: S317A (298 in EU numbering). Type 5

improves binding to  $\text{Fc}\gamma\text{RIIIA}$ , but does not affect binding to  $\text{Fc}\cdot\text{gamma}\cdot\text{RIIA}$  and  $\text{Fc}\gamma\text{RIIB}$ : E352A, K353A (333 and 334 in EU numbering).

DETD . . . of the  $\text{Fc}\gamma\text{RI}$ ,  $\text{Fc}\gamma\text{RII}$ , and  $\text{Fc}\gamma\text{RIII}$  subclasses, including allelic variants and alternatively spliced forms of these receptors.  $\text{Fc}\gamma\text{RII}$  receptors include  $\text{Fc}\cdot\text{gamma}\cdot\text{RIIA}$  (an "activating receptor") and  $\text{Fc}\gamma\text{RIIB}$  (an "inhibiting receptor"), which have similar amino acid sequences that differ primarily in the cytoplasmic domains thereof. Activating receptor  $\text{Fc}\cdot\text{gamma}\cdot\text{RIIA}$  contains an immunoreceptor tyrosine-based activation motif (ITAM) in its cytoplasmic domain. Inhibiting receptor  $\text{Fc}\gamma\text{RIIB}$  contains an immunoreceptor tyrosine-based inhibition motif.

DETD . . . affinity of the analyte for the receptor is relatively weak, e.g. in the micromolar range as is the case for  $\text{Fc}\cdot\text{gamma}\cdot\text{RIIA}$ ,  $\text{Fc}\gamma\text{RIIB}$ ,  $\text{Fc}\gamma\text{RIIIA}$  and  $\text{Fc}\gamma\text{RIIIB}$ . The method involves the formation of a molecular complex that has an improved avidity for the . . .

DETD This assay determines binding of an IgG Fc region to recombinant  $\text{Fc}\cdot\text{gamma}\cdot\text{RIIA}$ ,  $\text{Fc}\gamma\text{RIIB}$  and  $\text{Fc}\gamma\text{RIIIA}$   $\alpha$  subunits expressed as His6-glutathione S transferase (GST)-tagged fusion proteins. Since the affinity of the Fc region . . . anti-IgG in a standard ELISA format (Example 2 below). The affinity of the other members of the  $\text{Fc}\gamma\text{R}$  family, i.e.  $\text{Fc}\cdot\text{gamma}\cdot\text{RIIA}$ ,  $\text{Fc}\gamma\text{RIIB}$  and  $\text{Fc}\gamma\text{RIIIA}$  for IgG is however in the micromolar range and binding of monomeric IgG1 for these receptors can . . .

DETD . . . into larger molecular weight complexes via the chimeric IgE Fab:VEGF interaction. The E27 component of this complex binds to the  $\text{Fc}\cdot\text{gamma}\cdot\text{RIIA}$ ,  $\text{Fc}\gamma\text{RIIB}$  and  $\text{Fc}\gamma\text{RIIIA}$   $\alpha$  subunits with higher avidity to permit detection in an ELISA format.

DETD . . . to C1q and hence did not activate complement were examined for their ability to bind to the Fc receptors:  $\text{Fc}\gamma\text{RI}$ ,  $\text{Fc}\cdot\text{gamma}\cdot\text{RIIA}$ ,  $\text{Fc}\gamma\text{RIIB}$ ,  $\text{Fc}\gamma\text{RIIIA}$  and  $\text{FcRn}$ . This particular study was performed using a humanized anti-IgE antibody, an IgG1 antibody with these mutations.

DETD . . . the present study, the effect of mutating various Fc region residues of an IgG1 antibody with respect to binding  $\text{Fc}\gamma\text{RI}$ ,  $\text{Fc}\cdot\text{gamma}\cdot\text{RIIA}$ ,  $\text{Fc}\gamma\text{RIIB}$  and  $\text{Fc}\gamma\text{RIIIA}$  as well as  $\text{FcRn}$  was evaluated. Antibody variants with improved as well as diminished FcR binding were. . .

DETD Low Affinity  $\text{Fc}\gamma\text{R}$  Binding ELISAs:  $\text{Fc}\cdot\text{gamma}\cdot\text{RIIA}$ ,  $\text{Fc}\gamma\text{RIIB}$  and  $\text{Fc}\gamma\text{RIIIA}$  binding ELISAs were performed as described in Example 1 above, with detection of the stable hexamer (consisting. . .

DETD . . . additional variant, T307Q, also displayed improved  $\text{FcRn}$  binding compared to E27 parent antibody.

TABLE 6

CH2 DOMAIN VARIANTS

IG2	Res#EU	FcRn			Fc $\gamma$ RI			$\text{Fc}\cdot$		
	$\text{gamma}\cdot\text{RIIA}$	$\text{Fc}\gamma\text{RIIB}$	$\text{Fc}\gamma\text{RIIIA}$					n	mean	sd
	(Kabat)	mean	sd	n	mean	sd		n	mean	sd
	mean	sd	mean	sd						

REDUCED BINDING TO ALL. . .

DETD

TABLE 7

CH3 DOMAIN VARIANTS

IG2	Res#EU	FcRn			Fc $\gamma$ RI			$\text{Fc}\cdot$		
	$\text{gamma}\cdot\text{RIIA}$	$\text{Fc}\gamma\text{RIIB}$	$\text{Fc}\gamma\text{RIIIA}$					n	mean	sd
	(Kabat)	mean	sd	n	mean	sd		n	mean	sd
	mean	sd	mean	sd						

B1 K338(358)A 1.14 0.90.  
 DETD . . . and the FcR binding activity of those variants is summarized in the following table.

TABLE 8

NON-ALANINE VARIANTS

IG2	(Kabat)	Res#EU	FcRn	Fc $\gamma$ RI			Fc.			
		gamma.RIIA	Fc $\gamma$ RIIB	Fc $\gamma$ RIIIA	mean	sd	n	mean	sd	mean
		sd	mean	sd	mean	sd	n	mean	sd	mean
222	D249(262)E				0.97			0.99.		

DETD

TABLE 9

COMBINATION VARIANTS

IG2	(Kabat)	Res#EU	FcRn	Fc $\gamma$ RI			Fc.			
		gamma.RIIA	Fc $\gamma$ RIIB	Fc $\gamma$ RIIIA	mean	sd	n	mean	sd	mean
		sd	mean	sd	mean	sd	n	mean	sd	mean

96 S267(280)A 1.41 1.72.

DETD This study includes a complete mapping of human IgG1 for human Fc $\gamma$ RI, Fc $\gamma$ RIIA, Fc $\gamma$ RIIB, Fc $\gamma$ RIIIA, and FcRn. An alanine-scan of all amino acids in human IgG1 Fc (CH2 and CH3 domains) exposed to . . . Fc $\gamma$ RI and FcRn are high affinity receptors and monomeric IgG could be evaluated in the assays for these two receptors. Fc $\gamma$ RIIA, Fc $\gamma$ RIIB and Fc $\gamma$ RIIIA are low affinity receptors and required use of an immune complex. Hence, an ELISA-type assay was used for Fc $\gamma$ RIIA,

Fc $\gamma$ RIIB, and Fc $\gamma$ RIIIA, in which pre-formed hexamers, consisting of three anti-IgE E27 and three IgE molecules were bound to the . . . al., Growth Factors 7:53 (1992) and Kim et al. Nature 362:841 (1993)). VEGF:anti-VEGF multimers also bound to the low affinity Fc $\gamma$ RIIA and Fc $\gamma$ RIIIA (FIGS. 16A and 16B).

DETD . . . presence of specific allelic forms (reviewed in Lehrnbecher et al. Blood 94(12):4220-4232 (1999)). Several studies have investigated two forms of Fc $\gamma$ RIIA, R131 and H131, and their association with clinical outcomes (Hatta et al. Genes and Immunity 1:53-60 (1999); Yap et al. . . . et al. J. Clin. Invest. 100(5):1059-1070 (1997)). In this example, selected IgG variants were tested against both allelic forms of Fc $\gamma$ RIIA or Fc $\gamma$ RIIIA. Fc receptor binding assays were performed essentially as described in the above examples. However, for Fc $\gamma$ RIIIA-V158, both (a) . . . Fc $\gamma$ RIIIA-V158) were carried out. The results of these studies are summarized in Table 10 below.

TABLE 10

Binding of Variants to Fc $\gamma$ RIIA and Fc $\gamma$ RIIIA Polymorphic Receptors

IG2	(Kabat)	IgG Complex			IgG Complex			IgG Complex		
		IgG Complex	IgG Monomer	Res#EU	Fc $\gamma$ RIIA-R131	Fc $\gamma$ RIIA-H131	Fc $\gamma$ RIIIA-F158	Fc $\gamma$ RIIIA-V158	Fc $\gamma$ RIIIA-V158	Fc $\gamma$ RIIIA-V158
		n	mean	sd	n	mean	sd	n	mean	sd

11 T256(269)A.

DETD . . . to the Fc $\gamma$ RIIIA-F158 form also showed improved binding to the Fc $\gamma$ RIIIA-V158 form though the improvement was not as

pronounced. For **Fc.gamma.RIIA-R131** (used in assaying all variants) and **Fc.gamma.RIIA-H131**, the binding pattern of the selected IgG1 variants did show some distinct differences. S267(280)A, H268(281)A, and S267(280)A/H268(281)A exhibited improved binding to **Fc.gamma.RIIA-R131**, compared to native IgG1, but not to **Fc.gamma.RIIA-H131**. In contrast, S267(280)G showed improved binding to **Fc.gamma.RIIA-R131** but reduced binding to **Fc.gamma.RIIA-H131** (Table 10). Other variants bound similarly to both allelic **Fc.gamma.RIIA** forms: V305(324)A, T307(326)A, N315(324)A, K317(336)A, and K320(339)A.

=> s FC(w)gamma(w)RIIa (7a) receptor

50010 FC  
19577 FCS  
64972 FC  
(FC OR FCS)  
228507 GAMMA  
838 GAMMAS  
228578 GAMMA  
(GAMMA OR GAMMAS)  
252 RIIA  
1 RIIAS  
252 RIIA  
(RIIA OR RIIAS)  
119017 RECEPTOR  
86804 RECEPTORS  
135368 RECEPTOR  
(RECEPTOR OR RECEPTORS)  
L2 167 FC(W)GAMMA(W)RIIA (7A) RECEPTOR

=> s 12/clm and (inhibitor or antagonist or composition)/clm

4476 FC/CLM  
187 FCS/CLM  
4656 FC/CLM  
( (FC OR FCS)/CLM)  
30568 GAMMA/CLM  
36 GAMMAS/CLM  
30577 GAMMA/CLM  
( (GAMMA OR GAMMAS)/CLM)  
37 RIIA/CLM  
25401 RECEPTOR/CLM  
6442 RECEPTORS/CLM  
28510 RECEPTOR/CLM  
( (RECEPTOR OR RECEPTORS)/CLM)  
17 FC/CLM(W)GAMMA/CLM(W)RIIA/CLM (7A) RECEPTOR/CLM  
24666 INHIBITOR/CLM  
7871 INHIBITORS/CLM  
29823 INHIBITOR/CLM  
( (INHIBITOR OR INHIBITORS)/CLM)  
8781 ANTAGONIST/CLM  
2570 ANTAGONISTS/CLM  
10416 ANTAGONIST/CLM  
( (ANTAGONIST OR ANTAGONISTS)/CLM)  
338169 COMPOSITION/CLM  
20320 COMPOSITIONS/CLM  
343333 COMPOSITION/CLM  
( (COMPOSITION OR COMPOSITIONS)/CLM)  
L3 12 (FC/CLM(W)GAMMA/CLM(W)RIIA/CLM (7A) RECEPTOR/CLM) AND (INHIBITOR OR ANTAGONIST OR COMPOSITION)/CLM

=> d bib,kwic 1-12

L3 ANSWER 1 OF 12 USPATFULL on STN

AN 2005:204449 USPATFULL

TI Fcγriia transgenic animal model for autoimmune disease

IN Hogarth, Phillip Mark, Victoria, AUSTRALIA  
Mottram, Patricia Lesley, Victoria, AUSTRALIA  
Sardjono, Caroline Tan, West Java, INDONESIA  
PI US 2005177876 A1 20050811  
AI US 2003-517251 A1 20030606 (10)  
WO 2003-AU718 20030606  
PRAI AU 2003-2856 20020607  
AU 2003-2002950529 20020801  
DT Utility  
FS APPLICATION  
LREP SHERIDAN ROSS PC, 1560 BROADWAY, SUITE 1200, DENVER, CO, 80202, US  
CLMN Number of Claims: 42  
ECL Exemplary Claim: 1  
DRWN 14 Drawing Page(s)  
LN.CNT 1445  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
CLM What is claimed is:  
of: a) administering a compound to be screened to a non-human transgenic animal that has been modified to express human **Fc**.  
**gamma.RIIa receptor** such that the transgenic animal is susceptible to an autoimmune disease; and b) assessing the transgenic animal to determine if.  
of: a) administering a compound to be screened to a non-human transgenic animal that has been modified to express human **Fc**.  
**gamma.RIIa receptor** such that the transgenic animal is susceptible to an autoimmune disease; and b) assessing the transgenic animal to determine if.  
disease, the method comprising the steps of: a) administering a compound to be screened to a non-human cell expressing human **Fc** **gamma.RIIa receptor**, wherein the cell is derived from a non-human transgenic animal that has been modified to express human **Fc.gamma.RIIa receptor** such that the transgenic animal is susceptible to an autoimmune disease; and b) assessing the cell to determine if the.  
claim 1, wherein the non-human transgenic animal is resistant to collagen-induced arthritis prior to being modified to express the human **Fc.gamma.RIIa receptor**.  
transgenic animal is a transgenic mouse derived from the strains C57BL/6 and SJL that has been modified to express human **Fc**.  
**gamma.RIIa receptor**.  
21. A **composition** for treating or preventing an autoimmune disease, the **composition** comprising an effective amount of a compound that can reduce aberrant immune activity in an animal, and a pharmaceutically acceptable.  
22. A **composition** according to claim 21, wherein the compound can reduce aberrant immune complex formation, aberrant immune complex clearance or immune complex.  
23. A **composition** according to claim 21, wherein the compound can reduce aberrant immune activity in the animal by inhibiting the activity of.  
24. A **composition** according to claim 21, wherein the autoimmune disease is caused by aberrant immune complex formation, aberrant immune complex clearance or.  
25. A **composition** according to claim 21, wherein the autoimmune disease is selected from the group consisting of arthritis and systemic lupus erythematosus.  
26. A **composition** according to claim 21, wherein the autoimmune disease is rheumatoid arthritis (RA).  
27. A **composition** according to claim 21, wherein the autoimmune disease is collagen-induced arthritis (CIA).  
28. A non-human transgenic animal that has been modified to express human **Fc.gamma.RIIa receptor** such that the transgenic animal is susceptible to an autoimmune disease,

wherein the transgenic animal is resistant to collagen-induced arthritis prior to being modified to express the human **Fc.gamma.RIIa receptor**.

29, wherein the transgenic mouse is derived from the strains C57BL/6 and SJL that has been modified to express human **Fc.gamma.RIIa receptor**.

transgenic animal model for autoimmune disease, the method comprising the steps of: a) introducing a nucleic acid molecule encoding human **Fc.gamma.RIIa receptor** to a cell of a non-human embryo; b) transferring the embryo to a foster mother; and c) assessing the resultant. non-human transgenic embryo is resistant to collagen-induced arthritis prior to the introduction of a nucleic acid molecule encoding a human **Fc.gamma.RIIa receptor**.

transgenic animal is a transgenic mouse derived from the strains C57BL/6 and SJL that has been modified to express human **Fc.gamma.RIIa receptor**.

42. A method for producing a **composition** for treating or preventing an autoimmune disease, the method comprising: a) selecting the compound by the method according to claim 1; and b) formulating the compound with a pharmaceutically acceptable diluent, excipient or carrier to produce the **composition**.

L3 ANSWER 2 OF 12 USPATFULL on STN  
AN 2005:195779 USPATFULL  
TI Pecam-1 modulation  
IN Gibbins, Jonathan M, School of Animal and Microbial Sciences,, The University of Reading, Whiteknights House, Reading, UNITED KINGDOM RG6 6AJ

Cicmil, Milenko, Reading, UNITED KINGDOM

PI US 2005169920 A1 20050804

AI US 2003-500027 A1 20021217 (10)  
WO 2002-GB5730 20021217

PRAI GB 2003-130832 20011222

DT Utility

FS APPLICATION

LREP WORKMAN NYDEGGER, (F/K/A WORKMAN NYDEGGER & SEELEY), 60 EAST SOUTH TEMPLE, 1000 EAGLE GATE TOWER, SALT LAKE CITY, UT, 84111, US

CLMN Number of Claims: 19

ECL Exemplary Claim: 1-18

DRWN 7 Drawing Page(s)

LN.CNT 664

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

CLM What is claimed is:

wherein the activator is selected from the group comprising; a small molecule, an antibody, an antibody derivative, an agonist, an **antagonist**, a ligand, a DNA sequence, a complementary DNA sequence, an antisense DNA sequence, a probe, a protein sequence, a recombinant. **PECAM-1**, a catalyst, shear, oxidative stress, **Fc $\epsilon$ RI**, the high affinity receptor for **Fc $\epsilon$ RI**, an activated form of the high affinity **receptor Fc $\gamma$** .  
**gamma.RIIA**, **Fc $\gamma$ .RIIA**,  
, the low affinity **receptor for Fc $\gamma$** .  
**RIIA** and an activated form of the low affinity **receptor Fc $\gamma$ .RIIA**.

L3 ANSWER 3 OF 12 USPATFULL on STN  
AN 2005:63790 USPATFULL  
TI Optimized Fc variants and methods for their generation  
IN Lazar, Gregory Alan, Glendale, CA, UNITED STATES  
Chirino, Arthur J., Camarillo, CA, UNITED STATES  
Dang, Wei, Pasadena, CA, UNITED STATES

Desjarlais, John R., Pasadena, CA, UNITED STATES  
Doberstein, Stephen Kohl, Pasadena, CA, UNITED STATES  
Hayes, Robert J., Radnor, PA, UNITED STATES  
Karki, Sher Bahadur, Pasadena, CA, UNITED STATES  
Vafa, Omid, Monrovia, CA, UNITED STATES  
PA Xencor, Inc. (U.S. corporation)  
PI US 2005054832 A1 20050310  
AI US 2004-822231 A1 20040326 (10)  
RLI Continuation-in-part of Ser. No. US 2003-672280, filed on 26 Sep 2003,  
PENDING Continuation-in-part of Ser. No. US 2003-379392, filed on 3 Mar  
2003, PENDING  
PRAI US 2003-477839P 20030612 (60)  
US 2003-467606P 20030502 (60)  
US 2002-414433P 20020927 (60)  
US 2003-442301P 20030123 (60)

DT Utility  
FS APPLICATION  
LREP Robin M. Silva, Esq., Dorsey & Whitney LLP, Intellectual Property  
Department, Four Embarcadero Center, Suite 3400, San Francisco, CA,  
94111-4187

CLMN Number of Claims: 37

ECL Exemplary Claim: 1

DRWN 32 Drawing Page(s)

LN.CNT 8390

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

CLM What is claimed is:

1. wherein said Fc variant binds with greater affinity to human Fc $\gamma$ RI and Fc $\gamma$ RIIa, but exhibits unaltered affinity to a human **receptor** selected from the group consisting of Fc $\cdot$ gamma $\cdot$ RIIa, Fc $\gamma$ RIIb, and Fc $\gamma$ RIIc.

11. A variant protein according to claim 3, wherein said Fc variant binds with greater affinity to human Fc $\cdot$ gamma $\cdot$ RIIa, but exhibits unaltered affinity to a human **receptor** selected from the group consisting of Fc $\gamma$ RI, Fc $\gamma$ RIIb, Fc $\gamma$ RIIc, and F $\gamma$ RIIa.

36. A pharmaceutical **composition** comprising a variant protein according to claim 1 and a pharmaceutically acceptable carrier.

L3 ANSWER 4 OF 12 USPATFULL on STN

AN 2004:202937 USPATFULL

TI Treatment of patients with multiple sclerosis based on gene expression changes in central nervous system tissues

IN Dangond, Fernando, Newton, MA, UNITED STATES

Hwang, Daehee, Seattle, WA, UNITED STATES

Gullans, Steven R., Natick, MA, UNITED STATES

PI US 2004156826 A1 20040812

AI US 2003-670766 A1 20030925 (10)

PRAI US 2002-414219P 20020927 (60)

DT Utility

FS APPLICATION

LREP FULBRIGHT & JAWORSKI L.L.P., SUITE 2400, 600 CONGRESS AVENUE, AUSTIN, TX, 78701-3271

CLMN Number of Claims: 56

ECL Exemplary Claim: 1

DRWN 2 Drawing Page(s)

LN.CNT 7243

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

CLM What is claimed is:

1. A method for treating or preventing multiple sclerosis (MS) comprising administering to a subject with MS a **composition** that causes an increase in the activity or expression of at least one gene product selected from the group consisting of . . .

3. The method of claim 2, wherein the second MS therapy is given before the **composition**.

4. The method of claim 2, wherein the second MS therapy is given after the **composition**.

5. The method of claim 2, wherein the second MS therapy is given concurrent with the **composition**.

7. The method of claim 1, wherein the **composition** comprises peptide.

8. The method of claim 1, wherein the **composition** comprises a small molecule.

9. The method of claim 1, wherein the **composition** is an organo-pharmaceutical.

10. The method of claim 1, wherein the **composition** comprises an expression cassette comprising a nucleic acid encoding the selected gene product and a promoter active in eukaryotic cells, . . .

17. The method of claim 1, wherein said **composition** is administered intradermally, subcutaneously, intramuscularly, intraperitoneally, intravenously, intranasally, intraalveolarly, parenterally, intrathecally, intraparenchymally or intraperitoneally.

18. The method of claim 1, wherein said **composition** is administered to said mammal more than once.

19. The method of claim 1, wherein said **composition** is administered to said mammal in discrete repeated dosings.

claim 1 or 20, wherein at least one gene product comprises cyclin E (X95406), thymocyte antigen CD1a (M28825), serine protease **inhibitor** p19 (U71364), and skeletal muscle troponin T (M21984).

protein (NeuroD2) (U58681), H-neuro-d4 (U43843), Lim-Domain Transcription Factor Lim-1 (HG4318-HT4588), Cyclin D3 (M92287), Cyclin E1 (M74093), Cyclin G1 (X77794), Kinase **Inhibitor** P27kip1 Cyclin-Dependent (HG4258-HT4528), Cdk-**inhibitor** p57KIP2 (KIP2) (U22398), HsMcm6 (D84557), Retinoblastoma related protein (p107) (L14812), thymidylate synthase-inducer transcription factor LSF (U03494), 218 kD Mi-2 protein. . . heparan sulfate proteoglycan (X62515), Guanylate kinase (GUK1) (L76200), RAD23A homolog (AD000092), Mismatch repair protein (hMLH1) (AF001359), thymidylate kinase (CDC8) (L16991), **Inhibitor** of apoptosis protein 1 (U45878), Lysosome-associated membrane protein-2 (S79873), GM2 activator protein (X62078), alpha mannosidase (U37248), Alpha mannosidase II isozyme.

25. A method for treating or preventing multiple sclerosis (MS) comprising administering to a subject with MS a **composition** that causes a decrease in the activity level or expression of a gene product selected from the group consisting of. . .

27. The method of claim 26, wherein the second MS therapy is given before the **composition**.

28. The method of claim 26, wherein the second MS therapy is given after the **composition**.

29. The method of claim 26, wherein the second MS therapy is given concurrent with the **composition**.

31. The method of claim 25, wherein the **composition** comprises peptide.

32. The method of claim 25, wherein the **composition** comprises a small molecule.

33. The method of claim 25, wherein the **composition** is an organo-pharmaceutical.

34. The method of claim 25, wherein the **composition** comprises an expression cassette comprising a nucleic acid encoding an antisense construct or a ribozyme targeting the selected gene product, . . .

41. The method of claim 25, wherein said **composition** is administered intradermally, subcutaneously, intramuscularly, intraperitoneally, intravenously, intranasally, intraalveolarly, parenterally, intrathecally, intraparenchymally or intraperitoneally.

42. The method of claim 25, wherein said **composition** is administered to said mammal more than once.

43. The method of claim 25, wherein said **composition** is administered to said mammal in repeated discrete dosings.

. . . light chain protein 14.1 (Ig lambda chain related) (M34516), IGHAI from Ig germline H-chain G-E-A region A: gamma-3 5 (J00220), **Fc**-gamma-RIIA IgG Fc receptor class IIA (X68090), Fc Receptor Iib3 For IgG, Low Affinity (HG491-HT491), Ig-like transcript 2 (U82279), Ig Heavy Chain VdjC Regions (HG4458-HT4727), Ig J chain (M12759), . . . HSP70B' (X51757), Thromboxane synthase (M80647), Thromboxane A2 receptor (D38081), Thrombospondin 2 (HG896-HT896), Granulocyte colony-stimulating factor receptor (CSF3R) (M59820), Plasminogen activator **inhibitor** type 1 N-terminus (X04729), Autoimmune Antigen Thyroid Disease-Related Antigen (HG3578-HT3781), Integrin beta-5 subunit (X53002), Integrin beta 7 subunit (S80335), Neuronal PAS1 (NPAS1) (U77968), Prointerleukin 1 beta (X04500), Interleukin 1 receptor antagonist IRAP (X53296), R kappa B (U08191), Cathepsin C (X87212), Lymphocyte Antigen Hla-G3 (HG273-HT273), Lymph node homing receptor (M25280), Monocyte chemoattractant protein-4 precursor (MCP-4) (U46767), thymosin beta (D82345), Tissue **inhibitor** of metalloproteinase 4 (U76456), Pancreatic phospholipase A-2 (PLA-2) (M21056), Fetal brain adenylyl cyclase (L05500), Adenylyl cyclase (L21993), Guanine nucleotide-binding protein. . . (M36429), Transducin-like enhancer protein (TLE3) (M99438), Low-Mr GTP-binding protein (RAB31) (U59877), 43 kDa inositol polyphosphate 5-phosphatase (Z31695), RAB7 (X93499), Ras **Inhibitor** Inf (HG511-HT511), R-ras (M14949), RasGTPase activating protein (D78156), Clone 110298 (L43579), Rab GDI (D13988), RhoE=26 kda GTPase homolog (S82240), HSPDE4C1. . . (M37238), GTPase activating protein (rap1GAP) (M64788), RasGTPase activating protein (D78156), Ras-Specific Guanine Nucleotide-Releasing Factor (HG2510-HT2606), Ras-related protein Rab5b (X54871), Ras **inhibitor** (Rin1) (L36463), RIN protein (Y07565), Guanine Nucleotide-Binding protein Rap2 (HG1996-HT2044), Guanine nucleotide-binding protein (Gi) alpha subunit (M27543), Guanine nucleotide-binding protein. . . protein (X90872), E6-AP ubiquitin protein ligase 3A (AF002224), Ribosomal DNA repeating unit (U13369), Vacuolar proton ATPase subunit D (X71490), Inter-alpha-trypsin **inhibitor** subunit 3 (X16260), Leukemia virus receptor 1 (GLVR1) (L20859), Clone S171 (L40393), Clone cd24-1 Huntington's candidate region fragment (L37199), FLII. . . 1 (X68149), Trabecular meshwork-induced glucocorticoid response protein (AF001620), bHLH-PAS protein Jap3 (U60415), Guanylin (M97496), Dioxin-responsive (S81578), RD/X99296 (X99296), Plasma inter-alpha-trypsin **inhibitor** heavy chain H(3) (X14690), Major Yo paraneoplastic antigen (CDR2) (M63256), PCI (plasminogen activator **inhibitor** 3) from protein C **inhibitor** (M68516), KNP-1b; Also: U53003 (D86062), MJD1=MJD1 protein {CAG repeats} (S75313), POM121-like 1 (D87002), Cell surface glycoprotein P3.58 (M55024), Oviductal glycoprotein. . . Kallmann syndrome (KAL) (M97252), PACAP receptor (D17516), Retinal pigment epithelium-specific 61 kDa protein (RPE65) (U18991), Squamous cell carcinoma antigen-serine protease **inhibitor** (S66896), Clone 23948 sequence (U79293), Albumin, 3; Also: HG2841-HT2970, HG2841-HT2968 (HG2841-HT2969), Protein containing SH3 domain SH3GL2 (X99657), HK2 hexokinase II. . .

50. A method of identifying a **composition** useful in the treatment or prevention of multiple sclerosis (MS) comprising: (a)

providing a cell that expresses one or more. . . . or more genes, wherein modulation of the expression of the one or more genes identifies said candidate substances as a **composition** useful in the treatment or prevention of multiple sclerosis MS.

L3 ANSWER 5 OF 12 USPATFULL on STN  
AN 2004:64262 USPATFULL  
TI Methods of inhibiting phagocytosis  
IN Schreiber, Alan D., Philadelphia, PA, UNITED STATES  
PA Park, Jong-Gu, Drexel Hill, PA, UNITED STATES  
PI THE UNIVERSITY OF PENNSYLVANIA, Philadelphia, PA (U.S. corporation)  
AI US 2004048781 A1 20040311  
AI US 2003-639662 A1 20030813 (10)  
RLI Continuation of Ser. No. US 2001-811492, filed on 20 Mar 2001, GRANTED, Pat. No. US 6638764 Continuation of Ser. No. US 1998-158980, filed on 14 Sep 1998, GRANTED, Pat. No. US 6242427 Continuation of Ser. No. US 1996-657884, filed on 7 Jun 1996, GRANTED, Pat. No. US 5858981 Continuation-in-part of Ser. No. US 1995-483530, filed on 7 Jun 1995, ABANDONED Continuation-in-part of Ser. No. US 1994-316425, filed on 30 Sep 1994, ABANDONED Continuation-in-part of Ser. No. US 1993-129381, filed on 30 Sep 1993, ABANDONED

DT Utility  
FS APPLICATION  
LREP NIXON & VANDERHYE, PC, 1100 N GLEBE ROAD, 8TH FLOOR, ARLINGTON, VA, 22201-4714  
CLMN Number of Claims: 59  
ECL Exemplary Claim: 1  
DRWN 11 Drawing Page(s)  
LN.CNT 1469

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

CLM What is claimed is:

in a mammal comprising introducing into phagocytic cells of said mammal that are in contact with said immune complexes an **inhibitor** of a kinase endogenous to said cells associated with an Fc receptor present at the membrane of said cells, said. . . .  
3. The method according to claim 1 wherein said **inhibitor** is a peptide or mimetic.

20. The method according to claim 19 wherein said Fc receptor is Fc $\gamma$ RI, **Fc $\cdot$ gamma.RIIA** or Fc $\gamma$ RIIIA.

22. The method according to claim 21 wherein said Fc receptor is Fc $\gamma$ RI, **Fc $\cdot$ gamma.RIIA** or Fc $\gamma$ RIIIA.

the signal transduction of the  $\gamma$  subunit of the IgE receptor Fc $\epsilon$ RI comprising introducing into cells bearing said receptor an **inhibitor** of a kinase endogenous to said cells that activates said signal transduction of said Fc $\epsilon$ RI receptor or the  $\gamma$  subunit. . . .

31. The method according to claim 30 wherein said **inhibitor** is a peptide or mimetic.

51. The method according to claim 47 wherein said **inhibitor** targets the interval region between the second SH2 domain and catalytic (kinase) domain of Syk kinase.

59. A pharmaceutical **composition** comprising Syk kinase interval region, or portion thereof of at least 6 amino acids, or mimetic thereof, and a pharmaceutically . . . .

L3 ANSWER 6 OF 12 USPATFULL on STN  
AN 2003:112552 USPATFULL  
TI Use of bispecific antibodies to regulate immune responses  
IN Bigler, Michael Eric, Redwood City, CA, UNITED STATES  
Cherwinski, Holly Marie, Boulder Creek, CA, UNITED STATES

PI Phillips, Joseph H., Palo Alto, CA, UNITED STATES  
US 2003077282 A1 20030424  
AI US 2002-270084 A1 20021011 (10)  
PRAI US 2001-329182P 20011012 (60)  
DT Utility  
FS APPLICATION  
LREP DNAX RESEARCH, INC., LEGAL DEPARTMENT, 901 CALIFORNIA AVENUE, PALO ALTO,  
CA, 94304

CLMN Number of Claims: 17

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 1432

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

CLM What is claimed is:

5. The method of claim 1, wherein the activating receptor is selected from the group consisting of Fc $\epsilon$ RI, Fc $\gamma$ RIII, **Fc**.  
**gamma.RIIA**, Fc $\gamma$ RIIC, T-cell **receptor**,  
TREM-1, TREM-2, CD28, CD3, CD2, and DAP-12.

15. A **composition** comprising the bispecific antibody of claim 1 in conjunction with an acceptable carrier.

L3 ANSWER 7 OF 12 USPATFULL on STN

AN 2003:105832 USPATFULL

TI Genetic manipulation of phagocytes for modulation of antigen processing and the immune response therefrom

IN Albert, Matthew, New York, NY, UNITED STATES  
Birge, Raymond, New York, NY, UNITED STATES

PI US 2003072743 A1 20030417

AI US 2002-238213 A1 20020910 (10)

RLI Continuation of Ser. No. US 2000-565958, filed on 5 May 2000, ABANDONED

DT Utility

FS APPLICATION

LREP KLAUBER & JACKSON, 411 HACKENSACK AVENUE, HACKENSACK, NJ, 07601

CLMN Number of Claims: 143

ECL Exemplary Claim: 1

DRWN 12 Drawing Page(s)

LN.CNT 3105

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

CLM What is claimed is:

17. The method of claim 16 wherein said signaling domain derived from a member of the **Fc receptor** family is the Fc $\gamma$ RI,  
**Fc $\gamma$ RIIA**, Fc $\gamma$ RIIB, or  
Fc $\gamma$ RIII  $\alpha$ -chain.

34. The method of claim 33 wherein said signaling domain derived from a member of the **Fc receptor** family is FcR $\gamma$ I, **Fc**.  
**gamma.RIIA**, Fc $\gamma$ RIIB, or FcR $\gamma$ III  
 $\alpha$ -chain.

53. The method of claim 52 wherein said signaling domain derived from a member of the **Fc receptor** family is FcR $\gamma$ I, **Fc**.  
**gamma.RIIA**, Fc $\gamma$ RIIB, or FcR $\gamma$ III  
 $\alpha$ -chain.

cells or precursors thereof to apoptotic cells expressing said antigen in the presence of at least one of the following **compositions**: i) an agent capable of both facilitating cross-priming and maturing said dendritic cell; or ii) the combination of at least.

135. The method of claim 134 wherein said signaling domain derived from a member of the **Fc receptor** family is the Fc $\gamma$ RI,  
**Fc $\gamma$ RIIA**, Fc $\gamma$ RIIB, or  
Fc $\gamma$ RIII  $\alpha$ -chain.

139. The integrin receptor heterodimer of claim 138 wherein said signaling domain derived from a member of the **Fe receptor**

family is the Fc $\gamma$ RI, **Fc $\gamma$ .RIIA**,  
Fc $\gamma$ RIIB, or Fc $\gamma$ RIII  $\alpha$ -chain.

The integrin receptor chimeric  $\beta$  subunit of claim 141 wherein  
said signaling domain derived from a member of the **Fc receptor**  
family is the Fc $\gamma$ RI, **Fc $\gamma$ .RIIA**,  
Fc $\gamma$ RIIB, or Fc $\gamma$ RIII  $\alpha$ -chain.

L3 ANSWER 8 OF 12 USPATFULL on STN  
AN 2002:235026 USPATFULL  
TI Tripeptide of FcgammaRIIA  
IN Schreiber, Alan D., Philadelphia, PA, UNITED STATES  
Worth, Randall, Philadelphia, PA, UNITED STATES  
Petty, Howard R., Detroit, MI, UNITED STATES  
PI US 2002127209 A1 20020912  
AI US 2001-989298 A1 20011121 (9)  
PRAI US 2000-252460P 20001122 (60)  
DT Utility  
FS APPLICATION  
LREP Nixon & VANDERHYE P.C., 1100 North Glebe Road, 8th Floor, Arlington, VA,  
22201

CLMN Number of Claims: 25

ECL Exemplary Claim: 1

DRWN 5 Drawing Page(s)

LN.CNT 614

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

CLM What is claimed is:

4. The method according to claim 3 wherein said Fc receptor is  
**Fc $\gamma$ .RIIA** or a modified form thereof  
comprising at least 1 additional L-T-L peptide in the cytoplasmic domain  
thereof.

13. The method according to claim 11 wherein said agent is IFN- $\gamma$   
or an **inhibitor** of IL-4.

L3 ANSWER 9 OF 12 USPATFULL on STN  
AN 2002:133845 USPATFULL  
TI Methods of inhibiting phagocytosis  
IN Schreiber, Alan D., Philadelphia, PA, UNITED STATES  
Park, Jong-Gu, Drexel Hill, PA, UNITED STATES  
PA UNIVERSITY OF PENNSYLVANIA. (U.S. corporation)  
PI US 2002068703 A1 20020606  
US 6638764 B2 20031028  
AI US 2001-811492 A1 20010320 (9)  
RLI Continuation of Ser. No. US 1998-158980, filed on 14 Sep 1998, GRANTED,  
Pat. No. US 6242427 Continuation-in-part of Ser. No. US 1996-657884,  
filed on 7 Jun 1996, GRANTED, Pat. No. US 5858981 Continuation-in-part  
of Ser. No. US 1995-483530, filed on 7 Jun 1995, ABANDONED  
Continuation-in-part of Ser. No. US 1994-316425, filed on 30 Sep 1994,  
ABANDONED Continuation-in-part of Ser. No. US 1993-129381, filed on 30  
Sep 1993, ABANDONED

DT Utility

FS APPLICATION

LREP Nixon & Vanderhye P.C., 8th Floor, 1100 N. Glebe Rd., Arlington, VA,  
22201

CLMN Number of Claims: 59

ECL Exemplary Claim: 1

DRWN 11 Drawing Page(s)

LN.CNT 1477

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

CLM What is claimed is:

in a mammal comprising introducing into phagocytic cells of said  
mammal that are in contact with said immune complexes an  
**inhibitor** of a kinase endogenous to said cells associated with  
an Fc receptor present at the membrane of said cells, said.

3. The method according to claim 1 wherein said **inhibitor** is a

peptide or mimetic.

20. The method according to claim 19 wherein said **Fc receptor** is **Fc $\gamma$ RI**, **Fc $\gamma$ .RIIA** or **Fc $\gamma$ RIIIA**.

22. The method according to claim 21 wherein said **Fc receptor** is **Fc $\gamma$ RI**, **Fc $\gamma$ .RIIA** or **Fc $\gamma$ RIIIA**.

. the signal transduction of the  $\gamma$  subunit of the IgE receptor Fc $\epsilon$ RI comprising introducing into cells bearing said receptor an **inhibitor** of a kinase endogenous to said cells that activates said signal transduction of said Fc $\epsilon$ RI receptor or the  $\gamma$  subunit. . .

31. The method according to claim 30 wherein said **inhibitor** is a peptide or mimetic.

51. The method according to claim 47 wherein said **inhibitor** targets the interval region between the second SH2 domain and catalytic (kinase) domain of Syk kinase.

59. A pharmaceutical **composition** comprising Syk kinase interval region, or portion thereof of at least 6 amino acids, or mimetic thereof, and a pharmaceutically.

L3 ANSWER 10 OF 12 USPATFULL on STN  
AN 2002:119856 USPATFULL  
TI Fc receptor modulators and uses thereof  
IN Baell, Jonathan B., Ivanhoe, AUSTRALIA  
Garrett, Thomas P.J., Brunswick, AUSTRALIA  
Hogarth, P. Mark, Williamstown, AUSTRALIA  
Matthews, Barry R., Olinda, AUSTRALIA  
McCarthy, Thomas D., East Malvern, AUSTRALIA  
Pietersz, Geoffrey A., Greensborough, AUSTRALIA  
PA Ilexus Pty Limited (non-U.S. corporation)  
PI US 2002061844 A1 20020523  
US 6835753 B2 20041228  
AI US 2001-995277 A1 20011126 (9)  
RLI Continuation of Ser. No. US 1999-393598, filed on 10 Sep 1999, PENDING  
PRAI US 1998-99855P 19980911 (60)  
US 1999-131938P 19990430 (60)  
US 1999-148479P 19990811 (60)  
DT Utility  
FS APPLICATION  
LREP SHERIDAN ROSS PC, 1560 BROADWAY, SUITE 1200, DENVER, CO, 80202  
CLMN Number of Claims: 107  
ECL Exemplary Claim: 1  
DRWN 20 Drawing Page(s)  
LN.CNT 2100  
CLM What is claimed is:  
1. A pharmaceutical **composition** comprising: (a) compound selected from the group consisting of an aromatic compound of the formula: ##STR50## a heteroaromatic compound of. . .  
2. The **composition** of claim 1, wherein said compound is of the formula: ##STR55##  
3. The **composition** of claim 2, wherein said compound is of the formula: ##STR56##  
4. The **composition** of claim 3, wherein m and n are 0.  
5. The **composition** of claim 4, wherein W.sup.1 and W.sup.2 are CO<sub>2</sub>H.  
6. The **composition** of claim 5, wherein R.sup.1 and R.sup.2 are a bond.

7. The **composition** of claim 6, wherein L.sup.1 is --CH.sub.2CH.sub.2--.
8. The **composition** of claim 6, wherein L.sup.1 is --CH.sub.2O--.
9. The **composition** of claim 6, wherein L.sup.1 is --CH.dbd.CHC(.dbd.O)--.
10. The **composition** of claim 6, wherein L.sup.1 is --CH.sub.2CH.sub.2CH(OH)--.
11. The **composition** of claim 6, wherein L.sup.1 is --CH.dbd.CH--.
12. The **composition** of claim 6, wherein L.sup.1 is --CH(OH)CH(OH)--.
13. The **composition** of claim 12, wherein the stereochemistry of hydroxy groups is (S,S).
14. The **composition** of claim 6, wherein L.sup.1 is --CH.sub.2N(R.sup.26)CH.sub.2, wherein R.sup.26 is H, C.sub.1-C.sub.6alkyl or an amine protecting group.
15. The **composition** of claim 14, wherein R.sup.26 is --CH.sub.2CO.sub.2H.
16. The **composition** of claim 6, wherein L.sup.1 is a moiety of the formula: ##STR57##
17. The **composition** of claim 5, wherein R.sup.1 and R.sup.2 are --CH.sub.2--.
18. The **composition** of claim 17, wherein L.sup.1 is ethylene.
19. The **composition** of claim 17, wherein L.sup.1 is --CH.dbd.CH--.
20. The **composition** of claim 5, wherein R.sup.1 is methylene, R.sup.2 is a bond and L.sup.1 is ethylene.
21. The **composition** of claim 4, wherein W.sup.1 and W.sup.2 are PO(OR.sup.15).sub.2, and R.sup.1 and R.sup.2 are a bond.
22. The **composition** of claim 21, wherein L.sup.1 is ethylene.
23. The **composition** of claim 22, wherein R.sup.15 is ethyl.
24. The **composition** of claim 22, wherein R.sup.15 is H.
25. The **composition** of claim 21, wherein L.sup.1 is a moiety of the formula: ##STR58## wherein each of R.sup.27 and R.sup.28 is independently.
26. The **composition** of claim 25, wherein each of R.sup.27 and R.sup.28 is independently 4-methoxybenzyl or H.
27. The **composition** of claim 6, wherein L.sup.1 is a moiety of the formula: ##STR59## wherein each of R.sup.27 and R.sup.28 is independently.
28. The **composition** of claim 27, wherein each of R.sup.27 and R.sup.28 is independently 4-methoxybenzyl or H.
29. The **composition** of claim 4, wherein L.sup.1 is --CH.dbd.CH--, W.sub.1 and W.sub.2 are C(.dbd.NH)NH(OH), and R.sub.1 and R.sub.2 are bond.
30. The **composition** of claim 4, wherein L.sup.1 is

--CH<sub>2</sub>.sub.20--, W<sub>2</sub>.sub.1 and W<sub>2</sub>.sub.2 are C(.dbd.O)CF<sub>2</sub>.sub.3, and R<sub>2</sub>.sub.1 and R<sub>2</sub>.sub.2 are bond.

31. The **composition** of claim 4, wherein L<sub>2</sub>.sup.1 is --CH<sub>2</sub>CH<sub>2</sub>CH<sub>2</sub>--, R<sub>2</sub>.sub.1 and W<sub>2</sub>.sub.1 together forms --(CH<sub>2</sub>.sub.2).sub.aCH(NHR<sub>2</sub>.sup.29)CO<sub>2</sub>H, wherein a is an integer from 0. . .
32. The **composition** of claim 31, wherein R<sub>2</sub>.sub.2 and W<sub>2</sub>.sub.2 together forms --(CH<sub>2</sub>.sub.2).sub.bCH(NHR<sub>2</sub>.sup.30)CO<sub>2</sub>H, wherein b is an integer from 0 to 2 and. . .
33. The **composition** of claim 32, wherein a and b are 1, and R<sub>2</sub>.sup.29 and R<sub>2</sub>.sup.30 are --C(.dbd.O)CH<sub>2</sub>.sub.3.
34. The **composition** of claim 2, wherein said compound is of the formula: ##STR60##
35. The **composition** of claim 1, wherein said compound is of the formula: ##STR61##
36. The **composition** of claim 35, wherein said compound is of the formula: ##STR62##
37. The **composition** of claim 36, wherein Y<sub>2</sub>.sup.1 is --NH<sub>2</sub>.sub.2.
38. The **composition** of claim 37, wherein m and n are 0.
39. The **composition** of claim 1, wherein said compound is of the formula: ##STR63## wherein X<sub>2</sub>.sup.1, X<sub>2</sub>.sup.2, X<sub>2</sub>.sup.3 and X<sub>2</sub>.sup.4 are NR<sub>2</sub>.sup.16.
40. The **composition** of claim 39, wherein said compound is of the formula: ##STR64##
41. The **composition** of claim 1, wherein said compound is of the formula: ##STR65##
42. The **composition** of claim 1, wherein said compound is of the formula: ##STR66##
43. The **composition** of claim 42, wherein R<sub>2</sub>.sup.11 is lysine side chain residue, R<sub>2</sub>.sup.12 is 2'-phenylethyl and R<sub>2</sub>.sup.14 is --C(.dbd.O)CH<sub>2</sub>.sub.3.
46. The **method** of claim 45, wherein said **Fc receptor** is selected from the group consisting of **Fc<sub>2</sub>.gamma.** **RIIa**, **Fc<sub>2</sub>RIIb**, **Fc<sub>2</sub>RIIc** and mixtures thereof.

L3 ANSWER 11 OF 12 USPATFULL on STN  
AN 2002:51012 USPATFULL  
TI Fc receptor modulators and uses thereof  
IN Baell, Jonathan B., Ivanhoe, AUSTRALIA  
Garrett, Thomas P. J., Brunswick, AUSTRALIA  
Hogarth, P. Mark, Williamstown, AUSTRALIA  
Matthews, Barry R., Olinda, AUSTRALIA  
McCarthy, Thomas D., East Malvern, AUSTRALIA  
Pietersz, Geoffrey A., Greensborough, AUSTRALIA  
PA Ilexus Pty Limited, Victoria, AUSTRALIA (non-U.S. corporation)  
PI US 6355683 B1 20020312  
AI US 1999-393598 19990910 (9)  
PRAI US 1998-99855P 19980911 (60)  
US 1999-131938P 19990430 (60)  
US 1999-148479P 19990811 (60)  
DT Utility  
FS GRANTED  
EXNAM Primary Examiner: Spivack, Phyllis G.  
LREP Sheridan Ross P.C.

CLMN Number of Claims: 50  
ECL Exemplary Claim: 1  
DRWN 20 Drawing Figure(s); 20 Drawing Page(s)  
LN.CNT 1876  
CLM What is claimed is:  
1. A pharmaceutical **composition** comprising: (a) compound having the formula: ##STR50## or salts thereof, wherein each of W.sup.1 and W.sup.2 is independently CO.sub.2R.sup.3 having.  
2. The **composition** of claim 1, wherein said compound is of the formula: ##STR51##  
3. The **composition** of claim 2, wherein said compound is of the formula: ##STR52##  
4. The **composition** of claim 3, wherein W.sup.1 and W.sup.2 are CO.sub.2H.  
5. The **composition** of claim 4, wherein R.sup.1 and R.sup.2 are a bond.  
6. The **composition** of claim 5, wherein L.sup.1 is --CH.sub.2CH.sub.2--.  
7. The **composition** of claim 5, wherein L.sup.1 is --CH.dbd.CHC(.dbd.O)--.  
8. The **composition** of claim 5, wherein L.sup.1 is --CH.sub.2CH.sub.2CH(OH)--.  
9. The **composition** of claim 5, wherein L.sup.1 is --CH.dbd.CH--.  
10. The **composition** of claim 5, wherein L.sup.1 is --CH(OH)CH(OH)--.  
11. The **composition** of claim 10, wherein the stereochemistry of hydroxy groups is (S,S).  
12. The **composition** of claim 4, wherein R.sup.1 and R.sup.2 are --CH.sub.2--.  
13. The **composition** of claim 12, wherein L.sup.1 is ethylene.  
14. The **composition** of claim 12, wherein L.sup.1 is --CH.dbd.CH--.  
15. The **composition** of claim 4, wherein R.sup.1 is methylene, R.sup.2 is a bond and L.sup.1 is ethylene.  
16. The **composition** of claim 1, wherein the compound is of the formula: ##STR53##  
17. The **composition** of claim 1, wherein the compound is of the formula: ##STR54##  
18. The **composition** of claim 1, wherein the compound is of the formula: ##STR55##  
19. The **composition** of claim 1, wherein the compound is of the formula: ##STR56##  
20. The **composition** of claim 1, wherein the compound is of the formula: ##STR57##  
21. The **composition** of claim 1, wherein the compound is of the formula: ##STR58##  
22. The **composition** of claim 1, wherein the compound is of the

formula: ##STR59##

23. The **composition** of claim 1, wherein the compound is of the formula: ##STR60##

26. The method of claim 25, wherein said **Fc receptor** is selected from the group consisting of **Fc<sub>gamma</sub>**, **RIIa**, **Fc<sub>gamma</sub>RIIb**, **Fc<sub>gamma</sub>RIIc** and mixtures thereof.

L3 ANSWER 12 OF 12 USPATFULL on STN  
AN 1999:4639 USPATFULL  
TI Method of inhibiting phagocytosis  
IN Schreiber, Alan D., Philadelphia, PA, United States  
PA Park, Jong-Gu, Drexel Hill, PA, United States  
University of Pennsylvania, Philadelphia, PA, United States (U.S.  
corporation)  
PI US 5858981 19990112  
AI US 1996-657884 19960607 (8)  
RLI Continuation-in-part of Ser. No. US 1995-483530, filed on 7 Jun 1995,  
now abandoned which is a continuation-in-part of Ser. No. US  
1994-316425, filed on 30 Sep 1994, now abandoned which is a  
continuation-in-part of Ser. No. US 1993-129381, filed on 30 Sep 1993,  
now abandoned  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Saunders, David; Assistant Examiner: VanderVegt, F.  
Pierre  
LREP Nixon & Vanderhye P.C.  
CLMN Number of Claims: 29  
ECL Exemplary Claim: 1,7  
DRWN 21 Drawing Figure(s); 15 Drawing Page(s)  
LN.CNT 1687  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
CLM What is claimed is:  
1. A subunit of the IgE receptor Fc<sub>ε</sub>RI comprising  
introducing into lung cells of a mammal bearing said receptor a peptide  
**inhibitor** of Syk kinase, or a mimetic of said peptide, wherein  
said peptide comprises the sequence YXXL (SEQ ID NO:8), wherein.  
2. The method according to claim 1 wherein said **inhibitor** is  
said peptide.  
10. The method according to claim 1 wherein said **inhibitor**  
targets the interval region between the second SH2 domain and the  
catalytic (kinase) domain of Syk kinase.  
22. The method according to claim 21 wherein said **Fc receptor**  
is Fc<sub>γ</sub>RI, **Fc<sub>gamma</sub>.RIIa** or  
Fc<sub>γ</sub>RIIIA.  
24. The method according to claim 23 wherein said **Fc receptor**  
is Fc<sub>γ</sub>RI, **Fc<sub>gamma</sub>.RIIa** or  
Fc<sub>γ</sub>RIIIA.